REMARKS

Reconsideration and reexamination of this application are respectfully requested.

Claim 23 is amended and claims 78, 81-91, 94, 95, and 102 canceled. Claims 23, 26-34, 72, 73 76, 77, 103, and 104 are pending. Support for the claim amendment is found, for example, in Examples 8 and 9. No new matter is introduced.

Claim 31, 72, 73, 76, 77 103, and 104 are withdrawn. Applicant respectfully requests that those claims be rejoined and examined together with the other claims when generic claim 23 is allowed.

All of the claims stand rejected under 35 U.S.C. § 112, first paragraph, as allegedly lacking enablement. Action at Item 4. The basis for this rejection is the Examiner's feeling that methods of treating or *in vivo* inhibition of a Dengue virus infection are not enabled. Applicant respectfully disagree for the reasons of record. However, in order to expedite prosecution Applicant has amended claim 23 to recite a "method of inhibiting binding of a Dengue virus to a human cell, wherein the binding of the Dengue virus to the human cell is mediated at least in part by the binding of a Dengue virus effector molecule on the Dengue virus to one or more DC-SIGN receptor selected from DC-Specific ICAM-Grabbing Nonintegrin (DC-SIGN) and DC-Specific ICAM-Grabbing Nonintegrin Related (DC-SIGNR) expressed on the human cell." The method comprises "providing to the human cell a molecule that specifically binds to the DC-SIGN receptor . . . in an amount sufficient to inhibit the binding of the Dengue virus effector molecule to the DC-SIGN receptor to thereby inhibit binding of the Dengue virus to the human cell."

Applicant submits that that method is fully enabled by the specification in view of the knowledge of the skilled artisan when this application was filed. Specifically, Examples 8 and 9 show that a molecule that specifically binds to the DC-SIGN receptor (mannan and a DC-SIGN monoclonal antibody) inhibits binding of a Dengue virus effector molecule to the DC-SIGN receptor to thereby inhibit binding of the Dengue virus to a human cell. Based on Applicant's teaching a skilled artisan would have understood that routine experimentation could be used to practice the full scope of the claims. Accordingly, the claims are enabled and this rejection should be withdrawn.

Claims 29, 30, 84, and 85 stand rejected under 35 U.S.C. § 112, second paragraph, as allegedly indefinite. Action at 3. Claims 84 and 85 have been canceled, thus rendering this rejection moot as to those claims. Applicant respectfully submits that claims 29 and 30 are not indefinite.

Claims 29 recites that "the molecule that specifically binds to the DC-SIGN receptor comprises a binding moiety of the Dengue virus effector molecule, wherein the binding moiety specifically binds to the DC-SIGN receptor." Claim 30 recites that "the molecule that specifically binds to the DC-SIGN receptor comprises a binding moiety of the Dengue virus E glycoprotein, wherein the binding moiety specifically binds to the DC-SIGN receptor." The Examiner finds those claims indefinite because she finds it "unclear how the molecule that binds the DC-SIGN receptor is also a binding moiety of the Dengue virus effector molecule." The Examiner continued, asking: "[i]f the molecule that binds the DC-SIGN receptor is an antibody, then how is the antibody also a binding moiety of the Dengue envelope glycoprotein? Does the antibody bind both

the DC-SIGN receptor and the Dengue envelope glycoprotein? If so, then how can the antibody be a monoclonal antibody?"

In response, Applicant notes that claims 29 and 30 both specify that "the binding moiety specifically binds to the DC-SIGN receptor." That is consistent with the definition of "binding moiety" provided by the specification at paragraph 069, which states that a biding moiety is "that portion of a molecule that substantially retains the ability to bind to a second molecule when other portions of the molecule are removed or modified or when the binding moiety is placed into a heterologous context." The specification goes on to state that "in the case of an effector molecule as defined herein, a binding moiety of the effector molecule can be defined. A binding moiety of an effector molecule is that portion of the effector molecule that substantially retains the ability to bind to DC-SIGN when other portions of the molecule are removed or modified or when the binding moiety is placed into a heterologous context." The specification at paragraph 060 defines an effector molecule as "any molecule that specifically binds to the DC-SIGN receptor present on cells of a mammal."

Thus, "a binding moiety of the Dengue virus effector molecule," as recited by claim 29, is that portion of the Dengue virus effector molecule that substantially retains the ability to bind to DC-SIGN when other portions of the molecule are removed or modified or when the binding moiety is placed into a heterologous context. Because the effector molecule of claim 29 is a "Dengue virus effector molecule," it is a molecule present on Dengue virus. Thus, it is not an antibody.

In claim 30, "a binding moiety of the Dengue virus E glycoprotein," is that portion of the Dengue virus E glycoprotein (a Dengue virus effector molecule) that substantially

retains the ability to bind to DC-SIGN when other portions of the molecule are removed or modified or when the binding moiety is placed into a heterologous context. Thus, it is not an antibody.

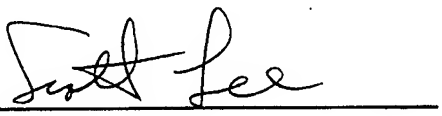
Applicant submits that claims 29 and 30 are definite and submits that this rejection should be withdrawn.

Please grant any extensions of time required to enter this response and charge any additional required fees to our Deposit Account No. 06-0916.

Respectfully submitted,

FINNEGAN, HENDERSON, FARABOW,
GARRETT & DUNNER, L.L.P.

Dated: July 6, 2007

By: 

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